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SHIP2 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP8473d

Specification

SHIP2 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession O15357

SHIP2 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 3636

Other Names
Phosphatidylinositol 3, 5-trisphosphate 5-phosphatase 2, Inositol polyphosphate phosphatase-like protein 1, INPPL-1, Protein 51C, SH2 domain-containing inositol 5'-phosphatase 2, SH2 domain-containing inositol phosphatase 2, SHIP-2, INPPL1, SHIP2

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP8473d was selected from the N-term region of human SHIP2. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

SHIP2 Antibody (N-term) Blocking Peptide - Protein Information

Name INPPL1 (HGNC:6080)

Function
Phosphatidylinositol (PtdIns) phosphatase that specifically hydrolyzes the 5-phosphate of phosphatidylinositol-3,4,5-trisphosphate (PtdIns(3,4,5)P3) to produce PtdIns(3,4)P2, thereby negatively regulating the PI3K (phosphoinositide 3-kinase) pathways (PubMed:16824732). Required for correct mitotic spindle orientation and therefore progression of mitosis (By similarity). Plays a central role in regulation of PI3K-dependent insulin signaling, although the precise molecular mechanisms and signaling pathways remain unclear (PubMed:9660833). While overexpression reduces both insulin-stimulated MAP kinase and Akt activation, its absence does not affect insulin signaling or GLUT4 trafficking (By similarity). Confers resistance to dietary

obesity (By similarity). May act by regulating AKT2, but not AKT1, phosphorylation at the plasma membrane (By similarity). Part of a signaling pathway that regulates actin cytoskeleton remodeling (PubMed:11739414, PubMed:12676785). Required for the maintenance and dynamic remodeling of actin structures as well as in endocytosis, having a major impact on ligand-induced EGFR internalization and degradation (PubMed:15668240). Participates in regulation of cortical and submembraneous actin by hydrolyzing PtdIns(3,4,5)P3 thereby regulating membrane ruffling (PubMed:21624956). Regulates cell adhesion and cell spreading (PubMed:12235291). Required for HGF-mediated lamellipodium formation, cell scattering and spreading (PubMed:15735664). Acts as a negative regulator of EPHA2 receptor endocytosis by inhibiting via PI3K-dependent Rac1 activation (PubMed:17135240). Acts as a regulator of neuritogenesis by regulating PtdIns(3,4,5)P3 level and is required to form an initial protrusive pattern, and later, maintain proper neurite outgrowth (By similarity). Acts as a negative regulator of the FC-gamma-RIIA receptor (FCGR2A) (PubMed:12690104). Mediates signaling from the FC-gamma-RIIB receptor (FCGR2B), playing a central role in terminating signal transduction from activating immune/hematopoietic cell receptor systems (PubMed:11016922). Involved in EGF signaling pathway (PubMed:11349134). Upon stimulation by EGF, it is recruited by EGFR and dephosphorylates PtdIns(3,4,5)P3 (PubMed:11349134). Plays a negative role in regulating the PI3K-PKB pathway, possibly by inhibiting PKB activity (PubMed:11349134). Down-regulates Fc-gamma-R-mediated phagocytosis in macrophages independently of INPP5D/SHIP1 (By similarity). In macrophages, down-regulates NF-kappa-B-dependent gene transcription by regulating macrophage colony-stimulating factor (M-CSF)-induced signaling (By similarity). Plays a role in the localization of AURKA and NEDD9/HEF1 to the basolateral membrane at interphase in polarized cysts, thereby mediates cell cycle homeostasis, cell polarization and cilia assembly (By similarity). Additionally promotion of cilia growth is also facilitated by hydrolysis of (PtdIns(3,4,5)P3) to PtdIns(3,4)P2 (By similarity). Promotes formation of apical membrane-initiation sites during the initial stages of lumen formation via Rho family-induced actin filament organization and CTNNB1 localization to cell-cell contacts (By similarity). May also hydrolyze PtdIns(1,3,4,5)P4, and could thus affect the levels of the higher inositol polyphosphates like InsP6. Involved in endochondral ossification (PubMed:23273569).

Cellular Location
Cytoplasm, cytosol. Cytoplasm, cytoskeleton. Membrane; Peripheral membrane protein. Cell projection, filopodium. Cell projection, lamellipodium. Basal cell membrane {ECO:0000250|UniProtKB:F1PNY0}. Nucleus {ECO:0000250|UniProtKB:D7PF45} Nucleus speckle {ECO:0000250|UniProtKB:D7PF45}. Cytoplasm, cytoskeleton, spindle pole {ECO:0000250|UniProtKB:F1PNY0} Note=Translocates to membrane ruffles when activated, translocation is probably due to different mechanisms depending on the stimulus and cell type (PubMed:11739414). Partly translocated via its SH2 domain which mediates interaction with tyrosine phosphorylated receptors such as the FC-gamma-RIIB receptor (FCGR2B). Tyrosine phosphorylation may also participate in membrane localization. Insulin specifically stimulates its redistribution from the cytosol to the plasma membrane. Recruited to the membrane following M-CSF stimulation. In activated spreading platelets, localizes with actin at filopodia, lamellipodia and the central actin ring.

Tissue Location
Widely expressed, most prominently in skeletal muscle, heart and brain. Present in platelets.

Expressed in transformed myeloid cells and in primary macrophages, but not in peripheral blood monocytes.

SHIP2 Antibody (N-term) Blocking Peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

SHIP2 Antibody (N-term) Blocking Peptide - Images

SHIP2 Antibody (N-term) Blocking Peptide - Background

SHIP2 (inositol polyphosphate-5 phosphatase-like 1), contains a phosphatase domain and an SH2 (src-homology domain 2) motif. SHIP2 iswidely expressed in fibroblast and nonhematopoietic tumor cell lines. Tyrosine phosphorylation of SHIP2 occurrs in response to treatment of cells with EGF, platelet-derived growth factor (PDGF), nerve growth factor (NGF), insulin-like growth factor-1 (IGF1), or insulin. EGF and PDGF induces transient tyrosine phosphorylation of SHIP2, while treatment of cells with NGF, IGF1, or insulin results in prolonged tyrosine phosphorylation of SHIP2, indicaating that SHIP2 may play a significant role in regulation of phosphatidylinositol 3-prime-kinase signaling by growth factors and insulin. Some animal models indicate that SHIP2 is a potent negative regulator of insulin signaling and insulin sensitivity in vivo, while others suggest that SHIP2 mediates obesity resistance but not changes in glucose and insulin homeostasis.

SHIP2 Antibody (N-term) Blocking Peptide - References

Koch, A., et al., Oncogene 24(21):3436-3447 (2005).Prasad, N.K., et al., J. Biol. Chem. 280(13):13129-13136 (2005).Kaisaki, P.J., et al., Diabetes 53(7):1900-1904 (2004).Tomlinson, M.G., et al., J. Biol. Chem. 279(53):55089-55096 (2004).Wang, Y., et al., J. Immunol. 173(11):6820-6830 (2004).